Worms, not germs

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Abstract

We present a case of a recent immigrant from India with 8 weeks of respiratory symptoms, eosinophilia, and diffuse reticulonodular opacity on chest X-ray. Further investigation revealed the cause to be tropical filarial pulmonary eosinophilia, for which he was successfully treated. We discuss the clinical features, investigation, and aetiology of this condition, which should be considered in patients from endemic areas.

We present a case of tropical filarial pulmonary eosinophilia.

Case report

A 27-year-old male, originally from India, was referred by his general practitioner to the Internal Medicine service with an 8-week duration of cough, producing occasional yellow sputum in the mornings. This was associated with chest pains, dyspnoea and wheezing. He had lost weight, but denied fevers or night sweats. Several courses of antibiotics were ineffective.

He denied any past medical history, in particular he had never suffered from asthma nor any other respiratory illnesses. He denied previous tuberculosis or known exposure. He was not taking regular medications.

He had been in New Zealand for 6 months. An immigration screening chest radiograph showed no evidence of pulmonary tuberculosis. He was a light smoker. Physical examination was normal, with no lymphadenopathy, chest signs, hepatosplenomegaly or skin changes. Initial investigations identified elevated white blood cell count with a marked eosinophilia at 8.6×10⁹/L. C-reactive protein was mildly elevated at 11 mg/L, and his electrolyte, renal and liver profile were all normal. The chest radiograph on presentation is shown in Figure 1.

Due to the suspicion of pulmonary tuberculosis, he was admitted into respiratory isolation. Sputum microscopy revealed eosinophils, but no acid-fast bacilli. Routine and mycobacterial cultures were negative. Serum IgE levels were grossly elevated at 2779 kU/L (normal range <100 kU/L). Aspergillus precipitins, Aspergillus-specific IgE enzyme allergosorbent testing, and serum anti-neutrophil cytoplasmic antibody were negative. Morning cortisol was within normal range. Stool microscopy was negative for ova, cysts and parasites. A peak flow diary did not demonstrate a diurnal pattern of bronchospasm. He was unable to perform lung function tests.

Serum Quantiferon Gold assay for Mycobacterium tuberculosis was “Positive”. He underwent a high-resolution CT scan of the lungs, shown in Figure 2.
Figure 1. Chest radiograph – diffuse reticulonodular opacity

Figure 2. High resolution CT scan of the chest – widespread miliary pattern of small nodules
Radiology reported a suspicion of miliary tuberculosis. On bronchoscopy the macroscopic appearance of the airways was normal, and multiple broncho-alveolar lavage samples were sent for analysis. These demonstrated 90% eosinophils, as shown in Figure 3. The samples were negative for acid-fast bacilli, parasites and malignant cells.

Figure 3. Cytology preparation of broncho-alveolar lavage – demonstrating 90% eosinophils

Serology for *Filaria* species was positive, with a signal-to-cutoff ratio of 10.0 (greater than 3.2 is considered positive). He received 2 mg/kg diethylcarbamazine thrice daily for 14 days, and on follow-up he reported a complete resolution of all symptoms. A repeat chest radiograph showed marked improvement. The diagnosis of *tropical filarial pulmonary eosinophilia* (TFPE) was therefore confirmed.

**Discussion**

TFPE is a clinical syndrome of paroxysmal non-productive cough, wheeze, occasionally weight loss, lymphadenopathy and low-grade fevers in association with
marked peripheral eosinophilia. Key laboratory features include markedly elevated serum IgE, high titre anti-filarial antibodies, and intense eosinophilia on bronchoalveolar lavage. Chest imaging usually reveals fine miliary lesions and increased bronchovascular markings. Lung function tests reveal a mixed pattern, and reversibility with bronchodilators.¹

The causative organisms are the mosquito-transmitted filarial helminth species, *Wuchereria bancrofti*, *Brugia malayi* or *Brugia timori*. These have overlapping distributions, and collectively they are endemic in most tropical regions. They cause lymphatic filariasis, which has numerous clinical manifestations, rarely including TFPE. The syndrome is characterised by a hypersensitivity response to the bloodborne micro-filariae, which become opsonised and deposited in lung and reticuloendothelial tissues. Pulmonary interstitial infiltrates result, eventually causing pulmonary fibrosis if untreated.²

Patients from tropical countries presenting with respiratory symptoms are often appropriately investigated for a suspicion of pulmonary tuberculosis. However, in this case the marked eosinophilia prompted a broader differential diagnosis, and investigations that led to the diagnosis and successful treatment of tropical filarial pulmonary eosinophilia.

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